nM P2 in both males and females, with more olgs in females than males in untreated and 2.5 nM P2. This difference was eliminated at 5 nM concentration. In contrast to P2, E2 did not have any effects on the number of olgs at 10 and 50 nM E2. However, at 100 and 500 nM concentrations, E2 increased cell death in enriched olgs cultures in both males and females. We found that glial cultures have large numbers of cell clusters resembling neurospheres but they have never been studied. The cultures derived from females had more clusters compared to males. These clusters were positive for NG2, an olg precursor marker, and to nestin, a marker found mainly in neuroepithelial multipotential cells. The number of clusters increased about 2 to 4× in the cultures treated with 10 and 50 nM E2 compared to controls in both males and females. This study shows E2 principally affects olg progenitor proliferation and not differentiation whereas; P2 mostly affects the olg differentiation.

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Misregulation of oligodendrocyte number in *inxs*^{vu56} zebrafish larvae

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The number and distribution of oligodendrocytes, the myelinating cell type of the vertebrate central nervous system (CNS), is regulated such that target axons have uniform myelination and rapid conduction of nerve impulses. The mechanisms that ensure specification of sufficient numbers of oligodendrocytes and their distribution throughout the nervous system are poorly understood. They arise as proliferative and migratory oligodendrocyte progenitors cells (OPCs) that are fated to give rise to oligodendrocytes from *olig2*+ neural precursors in the ventral spinal cord that also produce motor neurons. Near the end of embryogenesis, OPCs stop dividing and start differentiating into myelinating oligodendrocytes. We are attempting to elucidate mechanisms that control oligodendrocyte number and distribution using transgenic zebrafish that express green fluorescent protein under the control of the *olig2* promoter in a screen to identify genes that are necessary for oligodendrocyte development. One mutation, *inxs*^{vu56}, was identified because it produces excess dorsally positioned OPCs in the spinal cord. Based on antibody studies of neuronal markers, we determined that the excess does not appear to be the result of fate switching or global expansion of the CNS. Therefore, we hypothesize that the excess oligodendrocytes of *inxs*^{vu56} mutant larvae results from elevated levels of OPC proliferation. We are currently testing this hypothesis by examining proliferation markers and using time-lapse photomicroscopy.

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Divergent proliferative roles for Pax3 and Pax7 in chick

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Pax3 and Pax7 are closely related members of the paired-box transcription factor family that are important in muscle development in the vertebrate embryo. Evidence from a number of labs suggests that Pax3 and Pax7 are functionally redundant. Our lab previously showed that signals, such as Wnt3a, that maintain or induce Pax3 and Pax7 expression in the neural tube and dermomyotome also cause an increase in proliferation. Based on these findings, we hypothesized that Pax3 and Pax7 may function as positive regulators of proliferation. To determine if Pax3 and Pax7 have proliferative roles, we stained chick somite explants for endogenous Pax3, Pax7, and a proliferation marker, phosphohistone H3. Contrary to our expectations, we found that Pax3-positive cells were more likely to stain for H3 than Pax3-negative cells, and conversely Pax7-positive cells were less likely to stain for H3 than Pax7-negative cells. Overexpression of Pax3 in ovo resulted in an increase in the number of proliferative cells as compared to control while overexpression of Pax7 resulted in a decrease in the number of proliferative cells as compared to control. Cumulatively, our results strongly suggest different functional roles for Pax3 and Pax7 with respect to proliferation. We can envision at least two scenarios to explain why our results differ from previous studies. The effects of Pax3 and Pax7 on proliferation may be dose dependent or may be affected by the presence of alternative transcripts. We were able to identify alternative transcripts of both Pax3 and Pax7 in somites. Studies to evaluate the functional significance of these transcripts are pending.

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